CHAPTER

Intensive Care Management and Monitoring

David W. Newell and Arthur M. Lam

MONITORING AND MANAGEMENT OF INTRACRANIAL PRESSURE
- Concepts of Intracranial Pressure
- ICP Monitoring
- Characteristics of ICP Tracings
- Indications for ICP Monitoring
- Utility of ICP Measurements

TREATMENT OF INCREASED ICP
- Head Elevation
- Paralysis and Sedation
- Hyperventilation
- Diuretic Therapy
- Barbiturate Therapy for ICP Control
- Decompressive Craniectomy
- Other Agents Used to Lower ICP

THE USE OF STEROIDS IN HEAD INJURY

MONITORING OF CEREBRAL BLOOD FLOW

MONITORING OF JUGULAR VENOUS OXYGEN SATURATION

MONITORING OF EEG AND MULTIMODALITY-EVOKED POTENTIALS
- Monitoring of Somatosensory Evoked Potential
- Monitoring of Electroencephalogram

EXTRACRANIAL COMPLICATIONS IN HEAD-INJURED PATIENTS
- Hypotension
- Pneumonia
- Coagulopathy
- Sepsis

SEIZURE PROPHYLAXIS

CONCLUSIONS
Previous chapters have discussed much of the pathology and pathophysiology of acute head injury and the immediate objectives of its management. Equally important is the intensive care unit (ICU) management of those patients who did not require surgery and the postoperative management of those who had mass lesions removed. One should always remember the objectives of prevention of secondary injury and provision of optimal conditions for the recovery from primary brain injury. Intensive care management is geared toward providing optimal systemic support for cerebral energy metabolism and adequate cerebral perfusion for the injured brain. Many of the factors leading to recovery of primary brain injury are poorly understood, but it is evident that a significant component of the neurologic dysfunction that occurs after primary brain injury is reversible given the right conditions for recovery. Secondary brain injury can occur in the ICU in the form of hypoxia and ischemia from pulmonary complications, hypotension, or high intracranial pressure (ICP) from a variety of causes. The prompt recognition and treatment of these complications are essential in head injury management. This chapter will discuss the theoretical and practical aspects of intracranial pressure and its management as well as other aspects of monitoring and treatment of the head-injured patient.

MONITORING AND MANAGEMENT OF INTRACRANIAL PRESSURE

Concepts of Intracranial Pressure

The idea that abnormal intracranial pressure may occur and contribute to the pathogenesis of brain injury can be traced to the Monro-Kellie doctrine. This doctrine states that, because the brain and spinal cord are surrounded by inelastic coverings, changes in the volume of the contents would result in changes in the intracranial pressure. The volume of the intracranial contents consists of the brain tissue, blood, and cerebrospinal fluid. All of these components are relatively incompressible; therefore, an increase in one of the components or introduction of an expanding mass lesion will result in a decrease in the others so that the total volume is fixed. Increases in intracranial pressure as a result of increases in volume can occur according to the compliance of the system (see also Chap. 5).

The intracranial compliance can be defined as the change in intracranial volume divided by the change in intracranial pressure, or more appropriately, the intracranial elastance can be defined as the change in intracranial pressure divided by the change in intracranial volume. The intracranial compliance, or the ability of the system to absorb a change in volume, will be reduced as mass lesions enlarge, thereby reducing the amount of blood and CSF available to “buffer” the system. The intracranial compliance or elastance can be determined in patients with ventricular catheters in place. The slope of
the CSF volume versus pressure curve can be obtained by adding a known amount of fluid to the CSF through a ventricular catheter and noting the rise in pressure. Knowledge of the intracranial compliance can be helpful in head-injured patients by warning the physician of impending dangerous increases in ICP. The intracranial pressure wave form can also yield some information about the intracranial compliance. Although it is influenced by many factors including the recording method, the cardiac contractility, and the arterial pulse pressure, it usually has a higher pulse wave amplitude at a lower intracranial compliance (see Figs. 10-1 and 10-2).

ICP Monitoring

Continuous monitoring of intracranial pressure has become a standard practice in caring for patients with severe head injury. Various methods of monitoring are available and include epidural systems, subarachnoid bolts, and more recently, fiber-optic catheters that can be used in a subarachnoid bolt system or in a ventricular catheter (see Chap. 5). Ventriculostomy has always been considered the gold standard for ICP monitoring; however, fiber optic subarachnoid bolts compare very favorably. Computerized recording of the ICP wave forms with the capability for trend analysis has become standard in most modern intensive care units (see Fig. 10-1). Information gained from intracranial pressure monitoring can be used to guide ICP management as

![Figure 10-1](https://example.com/f101.gif) **Figure 10-1** Illustration of a short interval of continuous recording of ICP and arterial blood pressure. Notice the transmitted pulse wave in the ICP wave form due to transient cerebral blood volume changes (Scale = mmHg).
well as management of the cerebral perfusion pressure (CPP), which is the arterial blood pressure minus the intracranial pressure. Monitoring of ICP can also give early warning of expansion of mass lesions.

**Characteristics of ICP Tracings**

Measurements of intracranial pressure were first accomplished using the lumbar puncture and ventricular puncture methods. Continuous monitoring and recording of ICP was first reported by Guillaume and Janny and later by Lundberg. Lundberg published an extensive description of his findings during ICP monitoring in neurosurgical patients and carefully described the characteristics of the recordings as well as the influence of various pathologic conditions on the measurements. Lundberg observed various waves in the ICP tracings which he termed A-waves, B-waves, and C-waves.

A-waves or plateau waves, named because of their characteristic shape on the ICP tracing, are characterized by a sudden sharp increase in ICP to levels of 60 to 80 mmHg with a variable duration usually from 5 to 20 min. These waves often occurred in patients with large intracranial mass lesions, and their increasing frequency was a poor prognostic sign (see Fig. 10-3). Subsequently, it has been confirmed that an increase in cerebral blood volume and a decrease in cerebral blood flow (CBF) occurs during these waves. Rosner and coworkers have demonstrated in an experimental model that A-waves represent vasodilatation in response to a decrease in cerebral perfusion pressure. With reduced intracranial compliance this vasodilatation leads to in-
creases in ICP, which then further increases vasodilatation and a positive feedback mechanism is thus established. Treatment of A-waves includes increasing intracranial compliance, increasing cerebral perfusion pressure, and avoiding sudden stimuli that increase ICP or lower blood pressure.

B-waves described by Lundberg consist of repeating waves of variable amplitude in the ICP tracing, but usually between 10 and 20 mmHg. The frequency is also variable but is usually between 0.5 and 2 per minute. B-waves have been described in states of reduced intracranial compliance and are also believed to be due to fluctuations of intracranial blood volume. Figure 10-4 illustrates typical B-waves. Lundberg commented in his original description that they were associated with respiratory changes, and this has led to the widespread belief that they are due to CO₂ changes. He also observed the waves in ventilated patients, however, and stated that this observation permitted no definite conclusion as to their origins. Newell and coworkers demonstrated that continuous B-waves occurred in ventilated patients while recording end tidal CO₂. They clearly demonstrated that B-waves can occur despite a constant end tidal CO₂ concentration. They also demonstrated that the middle cerebral and extracranial internal carotid artery velocity, when measured using Doppler ultrasound, fluctuated synchronously with B-waves, and the amplitude of the waves was related to the ICP and the degree of velocity fluctuation. Velocity fluctuations at this frequency in the middle cerebral artery (MCA) were also demonstrated in normal volunteers. Therefore, there is a high probability that vasomotor waves causing fluctuations in cerebral blood flow can occur spontaneously at this frequency with concomi-

---

Figure 10-3 Example of a plateau wave or Lundberg A-wave⁶ in the intracranial pressure tracing. These waves are due to sudden increases in cerebral blood volume and cause decreased cerebral blood flow. Scale = mmHg
**Indications for ICP Monitoring**

Since the advent of continuous ICP monitoring, its most frequent use has been in head-injured patients. The true prevalence of high intracranial pressure after head injury and the pathophysiologic significance of this condition was not known until more experience was accumulated with the technique. ICP management has now been established as a critical component of the care of severely head-injured patients and it is useful to ensure an adequate CPP as

---

**Figure 10-4** Typical B-waves in the ICP tracing described by Lundberg. These waves reflect periodic fluctuation in cerebral blood volume due to vasomotor waves of the regulating vessels and usually indicate decreased intracranial compliance. Scale = mmHg

---

Significant fluctuations in cerebral blood volume. These become amplified in the ICP tracing when the intracranial compliance is reduced. The mechanism that controls the CBF fluctuations is unknown, but some authors suggest that a brainstem pacemaker may be responsible. Lundberg also described C-waves, which also may be due to reduced intracranial compliance reflecting small changes in intracranial blood volume in the ICP tracing. Lundberg suggested that these waves reflect arterial Traube-Hering waves and that their significance may be the indication of reduced intracranial compliance in some instances. Waves in the ICP tracing related to artificial respiration can also be seen, particularly if patients are hypovolemic. Figure 10-5 illustrates ICP waves produced by artificial respiration.
well as an early warning of the expansion of mass lesions. Studies have demonstrated a high prevalence of increased ICP after head injury in patients who have abnormal CT scans on admission to the hospital or in patients who are not following commands. ICP monitoring may also be indicated in patients with mild or moderate head injuries who require immediate general anesthesia for surgical treatment of systemic injuries.

Utility of ICP Measurements

Continuous monitoring of ICP following head injury has provided the essential information for guiding much of the therapy used to prevent secondary brain injury. It has also provided a rational basis to evaluate the effects of various modalities used to decrease ICP. Normally ICP should remain less than 10 mmHg, and usually treatment is initiated to decrease ICP following head injury when it is consistently more than 14 to 20 mmHg.

Increased ICP may produce secondary brain injury by several mechanisms. Increased pressure and pressure gradients caused by mass lesions may cause shifts in the position of brain subcomponents resulting in herniation. The classic herniations which have been described include (1) subfalcine, (2) uncal, (3) transtentorial, and (4) tonsillar. Each has specific clinical correlates and also specific patterns of pathologic damage that can occur as a result. A particular intracranial pressure that causes each of these entities cannot be specified, but more importantly, pressure gradients can occur that can result in herniation, even when the overall ICP is not extremely high. Brain herniation is often reversible after prompt recognition and treatment but may leave permanent injury to particular brain structures. For example, temporal lobe

![Graph showing ABP, ICP, and EtCO2 with respiration waves](image)

**Figure 10-5** Waves in the ICP tracing produced by secondary effects from artificial respiration. Scale = mmHg
masses can cause uncal herniation without markedly elevated ICP and should be treated if clinical signs exist.

Another major role of ICP monitoring is in helping to define the CPP and, thus, guide the therapy designed to ensure adequate cerebral perfusion. Autoregulation (the ability to maintain constant cerebral blood flow despite changing cerebral perfusion pressure) can be lost following head injury, and therefore, the brain is less protected from ischemia due to blood pressure fluctuations and increases in ICP.\textsuperscript{17,18} Decreases in arterial blood pressure can be as harmful as increased ICP in causing secondary brain ischemia. It is generally agreed that the cerebral perfusion pressure is optimal at a level greater than 70 mmHg; however, the exact optimal limits of CPP are controversial. A recent study has supported the concept that maintaining an adequate CPP is beneficial after head injury.\textsuperscript{19}

Continuous monitoring of ICP is also useful in providing early diagnosis of increases in the size of mass lesions. In patients who are unconscious, sedated or given paralytic agents, subtle changes in neurologic function are not always a sensitive indication of progressing brain injury. Progressive increases in ICP in these patients can prompt repeat CT scanning to detect surgically removable lesions that may be responsible for secondary deterioration in brain function.

Data are now available that clearly indicate that sustained elevation of ICP after head injury is associated with a poor prognosis.\textsuperscript{20,21} Marmarou and coworkers\textsuperscript{21} have recently reported data from the Traumatic Coma Data Bank that determined the relationship among increased ICP, hypotension, and outcome in patients with severe head injury. They demonstrated that the proportion of hourly ICP readings greater than 20 mmHg was highly significant in association with a poor outcome. The proportion of hourly blood pressure readings less than 60 mmHg was also highly significant in association with a poor outcome. Figure 10-6 illustrates these results.

Monitoring of ICP is now an integral part of head injury management and, despite some of the pitfalls that may be encountered, it provides valuable information for providing optimal care in the treatment of patients with head injury.

**TREATMENT OF INCREASED ICP**

There can be a variety of causes of increased intracranial pressure, and a number of treatments can be instituted depending on the cause. When ICP control becomes necessary, one must ensure that no new surgically correctable mass lesion has developed causing a progressive increase in ICP. Consideration must also be given to the maintenance of optimal conditions for ICP control and the accurate diagnosis of the causes of increased ICP to allow for rational treatment. Monitoring ICP using a ventricular catheter permits
Figure 10-6 Relationship between outcome and hypotension and ICP. Three-dimensional surface of estimated outcome probability versus the proportion of intracranial pressure (ICP) measurements greater than 20 mmHg \( [p(\text{ICP} > 20)] \) and the proportion of blood pressure measurements less than 80 mmHg \( [p(\text{BP} < 80)] \) for the vegetative/dead outcome group. To simplify the presentation, the other modeled factors were fixed at the following values: age = 30 years; admission motor score = 3 (flexion); and abnormal pupils = 1. The substantial effect of hypotension is readily evident from the front-to-back upward sloping of the surface. The impact of ICP elevation is apparent from the right-to-left upward sloping of the surface.

CSF drainage, which can be effective in controlling elevated ICP. Other strategies for ICP control are listed below.

**Head Elevation**

In order to maintain optimal conditions for cerebral venous return, the head of the bed should be elevated to 20 to 30° and one must ensure that a cervical collar or head rotation does not cause venous obstruction. The effect of head elevation on ICP has been well documented; however, there has been controversy as to the best head position for maintenance of optimal CPP.\(^{22-24}\) In some patients, particularly those who may be hypovolemic, raising the head of the bed can reduce the cerebral perfusion pressure.\(^{24}\) A recent study measured ICP, CPP, mean carotid pressure, and CBF at 0° and 30° angles in 22 head-injured patients. The results indicated that when the head of the
bed was elevated from 0 to 30°, no significant change in CPP or CBF occurred, but there was a significant reduction in ICP.23

Paralysis and Sedation

Excess agitation and movement can occur, especially in ventilated patients, making necessary the use of sedation and neuromuscular paralysis to help control ICP. Excess muscular activity, such as coughing or posturing and straining against the ventilator, can cause ICP to rise to abnormally high levels in patients who would otherwise have an acceptable ICP. Pancuronium bromide, or other shorter-acting agents, such as vecuronium and atracurium, can be given in moderate repeated doses to allow intermittent neurologic assessment. Intravenous lidocaine is also useful in suppression of cough and prevention of an increase in ICP associated with tracheal suctioning. Analgesics or narcotics can also be combined if paralysis alone is ineffective in decreasing ICP in agitated patients. Morphine given in small doses or a slow infusion can be useful and is also relatively rapidly reversible for neurologic assessment.

Hyperventilation

Hyperventilation can be a very effective way to rapidly reduce ICP. It is useful in acute situations and is routinely used in the initial stages of resuscitation of head-injured patients. The mechanism of action of hyperventilation is to cause generalized vasoconstriction primarily in the small regulatory arteries in the brain, which reduces the cerebral blood volume and reduces ICP. In patients with sudden increases in ICP due to expanding mass lesions, hyperventilation should be used to temporarily decrease ICP until more definitive therapy can be initiated. There are two aspects of the use of hyperventilation that are controversial: (1) The degree of hyperventilation that should be used, and (2) The importance of prolonged or chronic hyperventilation in head injury. One of the concerns over using vigorous hyperventilation is whether it can result in cerebral ischemia.25,26 It is recognized that autoregulation can be impaired or lost both focally and globally after head injury. Some authors suggest that hyperventilation can cause a "steal" of blood from poorly regulating brain areas and worsen focal ischemia. Moreover, vigorous manual "bagging" may also cause more generalized ischemia in certain patients.25-27

Concerns over prolonged or chronic hyperventilation stem from experimental observations that the vasoconstriction caused by decreasing PaCO2 is not sustained for long periods. Muizelaar28 demonstrated in rabbits that the observed pial artery vasoconstriction that occurs in response to hyperventilation begins to reverse after several hours and is gone at 24 h. If these experiments accurately reflect the situation in humans, they may indicate that there is a good rationale to use hyperventilation for short-term ICP control but
not for prolonged periods. A recent prospective randomized trial of hyperventilation in patients with severe head injury was completed and concluded that hyperventilation for 5 days was deleterious in those patients with motor scores on the Glasgow Coma Scale of 4 to 5. The outcome in these patients was significantly worse at 3 and 6 months, but no difference was observed at 12 months after injury. A third arm of the study by Muizelaar and coworkers included patients treated with hyperventilation and the buffer tromethamine (THAM). The addition of THAM reversed the deleterious effect of hyperventilation. The mechanism responsible for the poorer outcome at 3 and 6 months is unclear. There was no evidence of increased cerebral ischemia in the hyperventilated group. One possible factor may have been a rebound in ICP in the hyperventilated group. We try therefore, to keep the PaCO2 between 35 to 40 mmHg and to begin mannitol therapy as the next step for ICP control. Hyperventilation is used to cause a short-term decrease of ICP, e.g., during transport of the patient or CT scanning or during other situations when ICP can become transiently elevated. Efforts should be made to avoid prolonged continuous hyperventilation.

**Diuretic Therapy**

Reduction of ICP in head-injured patients can be accomplished effectively using osmotic diuretics. Mannitol has become the most commonly administered agent and is usually administered intravenously as a 20% or 25% solution. Doses commonly used range between 0.25 and 1.0 g/kg body weight. Mannitol may be used on a repeated schedule, but the serum osmolarity must be followed to ensure that systemic dehydration does not take place. Serum osmolarity should not be allowed to increase to more than 320 mosmol/kg. The mechanism of ICP reduction by mannitol may be related to its osmotic effect in shifting fluid from the brain tissue compartment to the intravascular compartment as well as its ability to improve blood rheology by decreasing blood viscosity. The latter effect causes vasoconstriction, which keeps blood flow constant when autoregulation is present, and can, therefore, reduce blood volume and ICP.

**Barbiturate Therapy for ICP Control**

On the basis of reports by Shapiro and coworkers indicating that barbiturates can lower ICP, barbiturate therapy has been used extensively in head-injured patients when other therapies, mostly hyperventilation and mannitol, failed to decrease ICP adequately. The mechanism of action of barbiturates is believed to be due to their ability to inhibit synaptic transmission, thereby decreasing the cerebral metabolic rate of oxygen consumption (CMRO2). In patients where metabolism and cerebral blood flow is coupled, decreased CMRO2 causes vasoconstriction, reducing blood volume and, thereby, de-
creasing ICP. Subsequent to the use of barbiturate therapy in head-injured patients, it was observed that some patients responded well with effective ICP control while others had a poor response. There are two major reasons why some patients may not respond to barbiturate therapy: One is because their $CMR_O_2$ may be already low because of their head injury and is not further decreased to any significant extent by barbiturates; the other is that cerebral blood flow and metabolism may be uncoupled. In this situation, even though $CMR_O_2$ can be decreased, vasoconstriction does not occur.

Several studies evaluating the effect of barbiturates on ICP control and outcome in head injury have been published. An improvement in the outcome could not be demonstrated in several early studies, and a significant prevalence of hypotension was seen. In these initial studies, barbiturates were given prophylactically to patients, and one study compared mannitol therapy with barbiturate therapy. Subsequently, a multicenter prospective study was performed in which those patients who failed conventional therapy to control ICP were to receive barbiturates or to continue conventional therapy. There was a 2:1 benefit in the barbiturate-treated group with respect to ICP control. This benefit was extended to 4:1 when patients were stratified by cardiac complications before random selection. In the treatment group, approximately one-third of the patients had their ICP controlled with barbiturates. An improvement in the outcome was also noted in those patients who responded as compared with the nonresponders.

Treatment with barbiturates requires a significant commitment of resources and personnel and is associated with a significant risk of complications. The most significant complication has been cardiac depression and instability, and therefore, it is generally accepted that patients with significant preexisting cardiac abnormality or instability should not undergo this therapy. Intensive monitoring is required including arterial blood pressure monitoring, pulmonary artery catheter monitoring, EEG monitoring, and frequent blood chemistries monitoring for barbiturate levels. In addition, intensive nursing assessment for pulmonary and thrombotic complications is required. Patients must be normovolemic, and hypovolemia and hypotension must be avoided. Barbiturate treatment is usually started with a slow intravenous infusion of pentobarbital, 5 to 10 mg/kg over 10 to 30 min while carefully observing the blood pressure. EEG monitoring is helpful to define the limits of therapy as indicated by the occurrence of burst suppression. Once burst suppression is achieved, then a constant infusion may be maintained. Serum levels should range between 30 and 50 mg/dl, and controlled burst suppression should be present on EEG.

**Decompressive Craniectomy**

In patients with refractory ICP increases, surgical decompressive craniectomy has been another treatment option. The practice of surgical decompression
of the bony covering of the brain and opening of the dura was practiced by Cushing, who performed subtemporal decompression for intracranial hypertension. Craniectomy for head injury has been performed using several different methods. The majority of the reports, however, have been in the pre-CT era. Kjellberg reported the technique of bifrontal craniectomy in patients with refractory intracranial hypertension from a variety of causes. Another method of circumferential craniectomy was reported on a small number of patients with poor results. The most common procedure used for cranial decompression has been hemicraniectomy or large unilateral cranial flaps with dural patching. Several series of patients undergoing hemicraniectomy for the treatment of hemispheric swelling associated with acute subdural hematoma have been reported. Reports indicate that this procedure can be effective in reducing ICP, but the exact role of the procedure has been controversial. We have used this procedure in patients with secondary deterioration and midline shift with high ICP, not controlled by medical therapy, and for intraoperative brain swelling after the removal of subdural hematomas (see Chap. 6).

Other Agents Used to Lower ICP

THAM

The buffer THAM has been used in experimental head injury as well as in several recent human trials. The mechanisms of action include correcting intracellular as well as CSF acidosis and causing vasoconstriction with reduction of cerebral blood volume. THAM has been shown to be effective in reducing ICP in two human studies. Wolf and coworkers demonstrated in a prospective randomized trial that there was no difference in outcome in the THAM-treated group; however, THAM was effective in decreasing ICP, and the THAM-treated group required less aggressive ICP treatment by other methods than the control group.

THE USE OF STEROIDS IN HEAD INJURY

High doses of steroids, mostly dexamethasone, have proven very effective in the treatment of brain swelling associated with tumors. Dexamethasone was also used frequently in the past to reduce brain edema and ICP in head-injured patients. Several large studies have now been performed indicating that steroids are not effective in reducing ICP and do not improve outcome in head-injured patients. Moreover, in patients with severe head injury who often have associated polytrauma, an increased risk of infections has been associated with steroid use. More recently, newer steroids, including methylprednisolone and 21-aminosteroids, which have a much lower glucocorticoid effect than dexamethasone, have been effective in experimental brain
and spinal cord injury and may act as antioxidants inhibiting free radical formation.\textsuperscript{59} Trials are currently underway to evaluate the use of these compounds following head injury. The 21-aminosteroid, Tirilazad, does not influence cerebral blood flow and the cerebrovascular response to CO\textsubscript{2} in healthy humans.\textsuperscript{60}

**MONITORING OF CEREBRAL BLOOD FLOW**

Cerebral blood flow measurements have yielded much important information about the pathophysiology of head injury. The exact involvement of CBF measurements in head-injured patients, however, remains controversial. The first practical method of obtaining CBF measurements was the Kety-Schmidt method using nitrous oxide washin. Subsequently, the xenon washout method became the most common technique, initially using intracarotid xenon injections, and subsequently using an intravenous delivery method.\textsuperscript{61} Portable systems are available that can be used in the intensive care unit. Other methods for evaluating cerebral blood flow in head-injured patients include thermal dilution probes,\textsuperscript{62} laser Doppler probes,\textsuperscript{63} transcranial Doppler,\textsuperscript{64} single photon emission computed tomography (SPECT), positron emission tomography (PET), and xenon-CT.\textsuperscript{65}

Most of the data on CBF after head injury have been gathered using the xenon washout method.\textsuperscript{61} This method can provide repeated single measurements of flow values in the vicinity of externally placed detectors. Langfitt and Obrist\textsuperscript{66} have determined several clinical uses of CBF measurements using this method in head-injured patients. These include (1) ensuring adequate CBF; (2) assessing outcome prediction; (3) evaluation of cerebrovascular responses, such as autoregulation and CO\textsubscript{2} reactivity, and (4) assessing the effects of treatments on CBF. It has been demonstrated that CBF can be normal, increased, or reduced after head injury. Obrist and coworkers\textsuperscript{61} have shown that increased CBF values usually indicate cerebral hyperemia and occur when there is uncoupling of CBF from cerebral metabolism. This condition is associated with high ICP. Low CBF after head injury has been associated with poor outcome.\textsuperscript{66}

Recently xenon-CT has been used to measure CBF much earlier during the hospitalization of head-injured patients than had previously been done using the external detector system.\textsuperscript{65} In order to evaluate the time interval in which cerebral ischemia can occur, the measurements in the study were made at the time of the admission CT scans. Bouma and coworkers\textsuperscript{65} reported that a significant number of patients demonstrated low CBF at this point in time, thus indicating early ischemia. In a group of 35 comatose head-injured patients, 31.4 percent demonstrated global or regional ischemia. Patients with diffuse swelling were especially prone to ischemia, which was associated with early mortality.
Laser Doppler and thermodilution probes have been used in neurosurgical patients to measure blood flow continuously.\textsuperscript{62,63} Continuous monitoring offers advantages over the single measurement methods; however, the probes must be implanted surgically and can only measure CBF in a very focal region. These drawbacks have limited the use of these techniques in head injury.

Transcranial Doppler has also been used to evaluate cerebral circulatory changes after head injury. By observing relative changes in blood flow velocity, blood flow changes can be evaluated under certain circumstances, and information about autoregulation and CO\textsubscript{2} reactivity can be collected.\textsuperscript{64-70} Chan and coworkers\textsuperscript{71} have reported a relationship between middle cerebral artery blood flow velocity and outcome in head-injured patients. Low blood flow velocity, probably indicating low CBF, correctly predicted 80 percent of the deaths. Low blood flow velocity with reversal of flow in diastole has also been demonstrated to indicate progressive impairment and arrest of the cerebral circulation (see Chaps. 5 and 11). Transcranial Doppler has also been useful in indicating posttraumatic vasospasm.\textsuperscript{72-74} Recent studies indicate that vasospasm has been underrecognized in head injury and may be associated with cerebral infarction.\textsuperscript{64,72-75} Weber and coworkers\textsuperscript{73} reported a 40 percent prevalence of vasospasm in head-injured patients using transcranial Doppler criteria.

Martin and coworkers\textsuperscript{74} have also reported a high prevalence of cerebral vasospasm using transcranial Doppler in head-injured patients. In a series of 30 head-injured patients, 27 percent developed vasospasm as confirmed by Doppler criteria. Three patients developed severe vasospasm confirmed by angiography, and one patient developed a cerebral infarction. Both of these studies discovered an association between posttraumatic vasospasm and subarachnoid hemorrhage. Posttraumatic vasospasm, which can easily be diagnosed with transcranial Doppler, must be considered as a cause of secondary deterioration in head-injured patients, especially in those with subarachnoid hemorrhage (see Figure 10-7).

**MONITORING OF JUGULAR VENOUS OXYGEN SATURATION**

Sampling of venous blood from the jugular bulb by using a retrograde jugular catheter can be performed to determine the jugular venous oxygen saturation (S\textsubscript{JvO\textsubscript{2}}). More recently, fiberoptic catheters have been used to continuously monitor the S\textsubscript{JvO\textsubscript{2}} in head-injured patients. Initial experience with this technique indicates that it can be performed with minimal complications and can provide useful information.\textsuperscript{76-79} One difficulty which has been encountered is interruption of the signal due to catheter movement. A strong positive correlation has been established between S\textsubscript{JvO\textsubscript{2}} measurements derived from the cooximeter catheter and S\textsubscript{JvO\textsubscript{2}} measurements derived from blood sampling.\textsuperscript{78} Sheinberg and coworkers\textsuperscript{78} reported the results of continuous S\textsubscript{JvO\textsubscript{2}} monitoring in 45 head-injured patients. They associated episodes of desaturation,
MONITORING OF EEG AND MULTIMODALITY-EVOKED POTENTIALS

Monitoring of Electroencephalogram

With the advent of modern imaging techniques, the electroencephalogram (EEG) no longer has any value as a diagnostic tool in patients suffering from head injury. Although various patterns (alpha coma, spindle coma, delta coma, burst suppression, electric silence, etc.) have been described, EEG monitoring does not materially alter the management of these patients.
Monitoring of Somatosensory-Evoked Potential

Monitoring of somatosensory-evoked potential (SSEP) in comatose patients for prognostic purposes was first introduced into clinical practice by Hume and Cant. They reported that the central conduction time (the time required for the impulse generated in response to a peripheral stimulus to travel from the brainstem to the primary sensory cortex; i.e., the difference in latency between the brainstem component and the cortical component) correctly predicted outcome in approximately 78 percent of patients suffering from traumatic coma. In contrast, monitoring of brainstem auditory-evoked potential (BAEP) was less sensitive but more specific; i.e., significant changes in BAEP represented rostral-caudal deterioration and inevitably led to death, whereas the presence of a normal BAEP did not necessarily result in good outcome. Houlden and coworkers reported that in 51 patients with head injuries, SSEP grades had a higher positive correlation with the Glasgow Outcome Score than GCS scores did. Conversely, Lindsay and coworkers reported that SSEP monitoring increased only slightly the predictive accuracy when compared with the clinical data alone. These authors concluded that evoked potential monitoring may be useful in paralyzed or sedated patients, but is not justified in patients where neurologic examination is feasible.

In some centers, multimodality-evoked potential monitoring including visual, somatosensory, and brainstem auditory potentials is used for prognostic purposes. In general, SSEP monitoring has the optimal correlation with outcome, and the addition of the other modalities improves the prognostic accuracy only marginally. More recently, motor-evoked potential (MEP) monitoring has been investigated as a prediction of outcome, however, the results are inconclusive. Although Facco and coworkers reported that the addition of MEP to SSEP monitoring improved the outcome prediction and decreased the rate of false negatives, Zentner and Rohde determined that MEP, in comparison with SSEP had no prognostic value. The value of evoked potential monitoring as a predictor of outcome in patients in coma and with severe head trauma was recently reviewed. Few studies have addressed the use of evoked potential monitoring in actual patient care management despite its demonstrated utility as a predictor of outcome. Moulten and coworkers performed automated continuous computerized monitoring of SSEP in 36 patients in posttraumatic coma and reported that, in addition to its use for outcome prediction, the management of three patients was altered as a result of SSEP monitoring. The authors conclude that continuous monitoring of SSEPs is a useful adjunct in the management of comatose head-injured patients. SSEP and BAEP usually resist the influence of intravenous anesthetic agents and remain recordable in patients treated with sedative agents, such as benzodiazepines and barbiturates, although the latencies may be increased and the amplitudes of the waveforms reduced. Of particular importance is the fact that SSEP and BAEP can be recorded in patients managed with therapeutic barbiturate coma in electrocortical si-
Evoked potential monitoring in these patients is valuable in providing the only physiological assessment of brain/brainstem function.

To summarize the literature, SSEP monitoring provides useful prediction in posttraumatic coma; the addition of BAEP monitoring provides only marginal benefit, and the use of MEP remains unproved. The use of evoked potential monitoring is not justified in patients where neurologic examination is feasible and, because of its technical complexity, its efficiency in improving patient management must also be considered unproved. It is, however, useful in the management of patients in therapeutic barbiturate coma. We currently do not use evoked potential monitoring routinely in the intensive care unit.

EXTRACRANIAL COMPLICATIONS IN HEAD-INJURED PATIENTS

Secondary injury to the brain can occur as a result of extracranial complications as well as from intracranial causes. Extracranial factors can interact and lead to cerebral damage as a result of infection, hypoxia, hypotension, coagulopathy, and other harmful conditions that can disturb overall homeostasis. The Traumatic Coma Data Bank\(^3\) analyzed the role of extracranial complications in determining the outcome in 734 severely head-injured patients. The most frequent complication was electrolyte disturbance (59 percent), but this did not appear to alter the outcome. Hypotension, pneumonia, coagulopathy, and sepsis were significant contributors to poor outcome and occurred frequently. Figure 10-8 shows the frequency of the major complications found in this study.

Hypotension

Hypotension, defined as a systolic blood pressure of less than 90 mmHg, can occur before or after hospitalization and can contribute to a poor outcome.\(^3\) Prehospital hypotension occurred in 34.6 percent of the Traumatic Coma Data Bank patients and doubled the mortality. Hypotension in the posthospitalization period occurred in between 20 and 25 percent of these patients. Elimination of this complication would have resulted in a 9.3 percent reduction in unfavorable outcome. The adequacy of the volume status of the patients must be ensured, and hypotension must be corrected rapidly if it occurs. Fluid restriction to reduce “brain swelling” does not appear justified, particularly if electrolytes are normal.

Pneumonia

Pneumonia is common in severely head-injured patients, occurring in 41 percent in two recent studies and usually several days after injury.\(^3,4\) The diagnosis can be established according to the following criterion: a new or
Figure 10-8 Incidence of extracranial complications from the Traumatic Coma Data Bank.93

progressing pulmonary infiltrate on chest roentgenograms in addition to two of the following findings: (1) new fever >38.5°C, (2) leukocyte count >12,000/mm³, and (3) new purulent tracheobronchial secretions.94

Pneumonia can prolong ICU and hospital stay.94 The Traumatic Coma Data Bank study estimated that a poor outcome could be avoided in 2.9 percent of all patients if pneumonia was eliminated as a complication.93

Coagulopathy

The coagulation abnormality that is most commonly encountered after head injury is disseminated intravascular coagulation (DIC).95 The pathogenesis of DIC can be directly due to brain injury when brain material, which is highly thrombogenic, enters the circulation. This condition is often associated with extensive basal skull fractures and dural sinus injury and may contribute to the pathogenesis of intracranial bleeding.96 Systemic injury can also cause DIC as a result of multisystem injury, hypothermia, massive transfusion, and fat embolism. The diagnosis of DIC is suggested by abnormally increased prothrombin time; partial thromboplastin time; and decreased platelets, plasma fibrinogen level, and hematocrit level; in addition to increased fibrin-split products. Confirmation of DIC can be obtained by using the plasma protamine test or ethanol gelation test.97–99 Treatment of DIC is reversal of the underlying cause. If the cause is brain injury alone, the process usually spontaneously reverses itself. The correction of systemic abnormalities, such
as hypothermia, shock, sepsis, and other primary causes will help to reverse the process. Blood components, such as platelets, fresh frozen plasma, red cells, and whole blood should be used as needed until the DIC resolves.

**Sepsis**

The prevalence of sepsis was approximately 10 percent in the group of patients studied by the Traumatic Coma Data Bank. It was estimated that 1.5 percent of poor outcomes could have been eliminated if sepsis had not occurred. Multiple sources of infection can occur in head-injured patients, particularly those with polytrauma, but special considerations should include the meticulous care of intravenous catheters and ventricular drains in addition to an awareness of sinus infections and meningitis.

**SEIZURE PROPHYLAXIS**

Seizures following head injury have been the topic of much study and controversy. Jennett and coworkers classified seizures into “early” and “late” and early seizures were considered to be the result of acute reactions to trauma. Conversely, late seizures are considered to be the result of formation of an epileptic focus, and, therefore, are considered to be posttraumatic epilepsy. Others have further categorized seizures into (1) immediate (occurring within 24 h after injury), (2) delayed early (occurring during the remainder of the first week, and (3) late (occurring more than 1 week after the trauma). The prevalence of delayed early seizures is estimated to be between 8 and 18 percent in patients with risk factors for seizures and between 18 and 48 percent for late seizures. Risk factors include GCS score <10, hematoma, contusion, penetrating injury, early seizures, and depressed skull fracture. A major controversy regarding posttraumatic seizures has been the involvement of prophylactic anticonvulsants. Previous studies have been inconclusive because of the study design and the failure to document adequate drug levels. A recent prospective randomized double blind study on phenytoin for posttraumatic seizures concluded that this drug reduced seizures during the first week after head injury but did not prevent the development of late seizures. We currently treat head-injured patients, who are at risk for seizures, with phenytoin after admission, maintain treatment for 1 week, and then discontinue the drug.

**CONCLUSIONS**

The ultimate outcome in the head-injured patient depends upon many factors. These include the premorbid condition of the patient, the severity of the
initial injury, and the effectiveness of the treatment on reversing and preventing further secondary injury, including the effects of complications and the length of the recovery. Effective intensive care management is essential in promoting the optimal recovery. Avoidance of secondary injury, particularly ischemia, is a primary consideration because of the high incidence of this complication. Of the various monitoring methods considered, each has its own unique capabilities, advantages, and disadvantages depending on the clinical situation. The recognition of secondary deterioration in head-injured patients requires the vigilance of the nursing staff and physicians who must rely on clinical judgment as well as the results of monitoring information and repeated radiographic studies. Prompt treatment of secondary deterioration is essential. Newer medications that provide cerebral protection against ischemia and brain injury are undergoing intensive investigation and clinical trials. The many factors which may promote neural recovery are also being investigated. In the future there will, no doubt, be many new clinical trials to evaluate these newer treatments.

REFERENCES


